REMARKS

In view of the above amendments and the following remarks, reconsideration of the outstanding office action is respectfully requested.

The prior application information has been included in response to paragraph 3 of the outstanding office action in the substitute specification.

A substitute specification is submitted herewith (paper copy and on disk) which includes sequence information. In accordance with 37 C.F.R. 1.821(f), this statement confirms that the contents of the Sequence Listing appearing on pages 1-26 (as submitted herewith) and on the computer readable 3.5" diskette (as submitted herewith) are the same and include no new matter.

The substitute specification includes an amended title.

The objection to claims 44 and 48 under 37 CFR 1.75(c) is hereby traversed in view of the above amendments.

The rejection of claims 1, 3, 6-10, 44 and 48 under 35 U.S.C. §112(first paragraph) for lack of written description is respectfully traversed.

The specification as filed satisfies the written description requirement for the claims. In particular, phosphorylation and activation of SGK by PDK1 is illustrated in Figures 4 and 17. Phosphorylation and activation of wild type SGK by PDK1 is discussed in the present specification at page 69, lines 13-25. Novel isoforms of SGK are discussed, for example, at page 82, line 12 to page 99, line 8 of the present application and the phosphorylation and activation of the isoforms by PDK1 was described in the application as filed. A description of SGK is found at page 6, line 5 to page 7, line 32, of the present application and includes, for example, human SGK, rat SGK, nematode SGK, and polypeptides

termed SGK2α, SGKβ and SGK3 (the sequences of which are disclosed). In addition, page 14, lines 12-27 discuss SGK fragments and fusions. Page 11, lines 1-7 of the present application as filed details where SGK includes SEQ ID NO:45 and/or SEQ ID NO:48. Phosphorylation of SGK by a preparation having PDK2 activity is disclosed in the application at page 19, lines 21-27 and in Example 1. Lastly, PDK1 polypeptides are described, for example, on page 16, lines 7-18 and page 18, lines 21-25 and in cited references therein for the preparation of fragments, variants and fusions of PDK1 that retain the required protein kinase activity.

With respect to claims 7-9 and written description of residues equivalent to Thr256 and Ser422 of full length human SGK1, as discussed on page 19, lines 14-15, the sequence for human SGK1 is known and disclosed, for example, in the cited reference. As discussed in Example 5 and on page 15, lines 11-14, for example, the residue equivalent to serine 422 of SGK1 in SGKα is serine 356 and in SGK3 is serine 419. The residue equivalent to threonine 256 of SGK1 in SGK2α is threonine 193 and in SGK3 is threonine 253. In addition, pages 18, line 6-page 19, line 19 of the specification as filed contains a discussion of how residues equivalent to Thr256, for example, can be identified.

To satisfy the written description requirement, a patent specification must describe the claimed invention in sufficient detail that one skilled in the art can reasonably conclude that the inventor had possession of the invention at the time the application was filed. Satisfactory disclosure depends on whether one of ordinary skill in the art would recognize that the applicant was in possession of the necessary common attributes or features of the elements possessed by the members of the genus in view of the species disclosed. In the present patent application, as filed, the claimed invention is described in sufficient detail to allow

one of ordinary skill in the art to recognize that applicants had possession of the claimed invention Accordingly, the rejection of the claims for lack of written description is improper and should be withdrawn.

The rejection of claims 1, 3, 6-10, 44 and 48 under 35 U.S.C. §112(first paragraph) for lack of enablement is respectfully traversed. In order for claims to be enabled, the specification, when filed, must contain sufficient information as to enable one skilled in the art to make and use the claimed invention. (Manual of Patent Examining Procedure ("MPEP") 2164.01). As long as the specification discloses at least on method for making and using the claimed invention that bears a reasonable correlation to the entire scope of the claim, the enablement requirement is satisfied. (In re Fisher, 427 F.2d. 833, 839, 166 USPQ 18, 24 (CCPA 1970); MPEP 2164.01(b)). In determining whether a patent application is in compliance with the enablement requirement, the PTO will consider whether one of ordinary skill in the art could practice the invention without undue experimentation. In re Wands, 858 F.2d. 731, 8 USPQ2d 1400 (Fed. Cir. 1988)).

In the present application, as discussed above in response to the written description rejection of the claims, the specification as filed includes examples and disclosure of all aspects of the invention as claimed. In particular, phosphorylation and activation of SDK (and isoforms thereof) by PDK1 are discussed (Example 1 and Example 5, for example). Phosphorylation by preparations which include PDK2 activity is shown (Example 1). A discussion of the phosphorylation of SGK isoforms and the phosphorylation sites are discussed throughout the application, for example, page 91, line 6-page 93, line 6. Accordingly, the rejection is improper and should be withdrawn.

The rejection of claims 6, 7-9, 44 and 48 under 35 U.S.C. §112(second paragraph) are respectfully traversed in view of the above amendments.

The rejection of claims 6 and 48 under 35 U.S.C. §101 is respectfully traversed in view of the above amendments.

The rejection of claims 1, 3, 6-10, 44 and 48 under 35 U.S.C. §103(a) for obviousness over Alessi et al., Current Biology 8:69-81(1997) ("Alessi") further in view of Waldegger et al. PNAS, USA 94(9):4440-4445(1997) ("Waldegger") is respectfully traversed.

Alessi relates to PDK1 and its role in the phosphorylation and activation of the p70 S6 kinase and PKB. Alessi does not teach or suggest activation and/or phosphorylation of SGK. Further, Alessi does not teach or suggest that PDK1 is capable of phosphorylating all serine/threonine protein kinases, in fact Alessi does not teach or suggest phosphorylation of any other kinases, other than the p70 S6 kinase and PKB.

Waldegger relates to a gene for a putative serine/threonine kinase, h-sgk, which has 98% sequence identity with a serum and glucocorticoid regulated kinase, SGK. There is no teaching or suggestion in Waldegger that a SGK is capable of being phosphorylated and activated, much less activated by PDK1.

Firstly, Alessi and Waldegger are not properly combinable. One skilled in the art of Alessi, which relates to phosphorylation of p70 S6 kinase by PDK1, would not look to Waldegger for guidance as Waldegger does not relate to phosphorylation. Likewise, one skilled in the art of Waldegger, which relates to a human skg gene, would not look to Alessi for guidance. There must be some suggestion in the references themselves, or in the state of the art, to combine the references (MPEP 2143.01 and 2145). Here, there is no such

suggestion. Accordingly, the combination of Alessi and Waldegger is improper and the rejection based on this combination should be withdrawn. There must be some suggestion in the references themselves, or in the state of the art, to combine the references. Here, there is no such suggestion.

Further, even assuming that the combination is proper, which it is not, the cited combination does not teach or suggest the present invention. As discussed above, neither Alessi or Waldegger, or the combination thereof, teach or suggest that SDK could be regulated by phosphorylation. In the Conclusions section of Alessi cited by the U.S. Patent and Trademark Office, Alessi teaches that "PDK1 is one of the components of the signaling pathway recruited by PI 3-kinase for the activation of p70 S6 kinase as well as of PKB, and serves as a multifunctional effector downstream of the PI 3-kinase." It is the PI 3-kinase pathway that is being considered in Alessi. Thus, Alessi does not teach or suggest phosphorylation of other serine/threonine kinases. Waldegger does not overcome this deficiency. As discussed above, there is nothing in Waldegger which teaches or suggest phosphorylation. In fact, Waldegger exclusively teaches that SGK is transcriptionally regulated (see, for example full paragraph on page 4440 preceding the Materials and Methods section, page 4444, first paragraph of Discussion section).

On page 10 of the outstanding office action, it is the PTO's position that "it would have been obvious to one having ordinary skill in the art at the time the invention was made to use the existing method of the Alessi et al . . . by substituting a human SKG . . . for p70, and do so with a reasonable expectation of success." It appears that the PTO is applying an impermissible "obvious to try" standard (MPEP 2145 (X)(B)). Neither Alessi or Waldegger give any indication

that SGK could be phosphorylated and the PTO's statement that the references suggest such a modification is incorrect.

Accordingly, the rejection based on the combination of Alessi and Waldegger is improper and should be withdrawn.

Pursuant to 37 CFR 1.97-1.98, applicants hereby submit the references listed on the enclosed PTO-1449 form. Also enclosed is a fee pursuant to 37 CFR 1.17(p).

In view of the foregoing, applicants submit that this case is in condition for allowance and such allowance is earnestly solicited.

Respectfully submitted,

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Date

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I hereby certify that this document is being deposited with the U.S. Postal Service as first class mail on 6/28/05 under 37 CFR 1.8 and is addressed to the Commissioner for Patent, PO Box 1450, Alexandria, VA 22313-1450

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